CASE REPORT JSLS

Laparoscopic Heller Myotomy With Epiphrenic Diverticulectomy

Timothy J. Pitchford, MD, Phillip D. Price, MD

ABSTRACT

Background and Objectives: To describe the technique and results of laparoscopic Heller myotomy and Toupet fundoplication combined with epiphrenic diverticulectomy.

Case Report: A 75-year-old man presented to our institution complaining of dysphagia to solid foods and liquids. The preoperative preparation included a barium swallow, esophagoscopy, and esophageal manometry. Three months earlier, the patient had a botulinum toxin injection, which provided temporary relief. Ten months later, the patient underwent a laparoscopic Heller myotomy and Toupet fundoplication combined with an epiphrenic diverticulectomy.

Results: No complications occurred. The patient tolerated clear liquids on postoperative day 1; on postoperative day 2, he was discharged tolerating full liquids. He returned to full activity in 1 week.

Conclusions: Epiphrenic diverticulectomy combined with treatment for the underlying motor disorder and gastroesophageal reflux prevention is an accepted practice. We demonstrate that this rare problem can be approached with the laparoscopic technique. Given this favorable result, we plan to continue this technique and establish a longer follow-up and wider series.

Key Words: Laparoscopy, Esophageal diverticulum, Achalasia.

Department of Surgery, Mount Carmel Health System, Columbus, Ohio, USA (all authors).

Address reprint requests to: Timothy J. Pitchford, MD, Marshfield Clinic, 2116 Craig Rd, Eau Claire, WI 54701, USA. Telephone: 715 858 4747, Fax: 715 858 4505

INTRODUCTION

Epiphrenic esophageal diverticula were first described in 1804 by Desguise as a mucosal protrusion between the muscular fibers of the esophagus.[1] Epiphrenic diverticula are categorized as a pulsion type of diverticulum. As suggested by its name, a pulsion diverticulum is usually found proximal to an area of high pressure. Even though achalasia is characterized by high pressure at the lower esophageal sphincter, the coexistence of an epiphrenic diverticulum is rare. In fact, esophageal diverticula are found more commonly with a nonspecific esophageal motility disorder or diffuse esophageal spasm.[2,3] This report describes the laparoscopic management of this rare disorder.

CASE REPORT

A 75-year-old man presented to the gastroenterologist with a history of regurgitation of old food with dysphagia to both liquids and solid food. Endoscopy revealed a considerable amount of retained food within the esophagus. Once this was cleared, a large diverticulum of the lower esophagus could be seen. Resistance was encountered when traversing the esophagogastric junction; however, no masses could be seen in the esophagus or stomach. Because of the resistance encountered, a 14-mm Savary-Gilliard dilator was passed to provide the patient with temporary relief. A follow-up barium esophogram showed a 13-cm x 11-cm esophageal diverticulum approximately 4 cm above the gastroesophageal (GE) junction projecting into the right hemithorax **(Figure 1)**. An esophageal manometry revealed a lower esophageal sphincter pressure of 15 mm Hg to 22 mm Hg. However, a high-pressure zone was encountered that measured 50 mm Hg to 100 mm Hg just distal to the esophageal diverticulum. Low primary peristaltic waves in the esophageal body were measured at 30 mm Hg during swallowing but occasionally reached 50 mm Hg to 60 mm Hg. The patient subsequently had 100 units of BoTox® (Allergan, Inc, Irvine, CA) injected into the lower esophageal sphincter (LES) providing complete symptomatic relief. Ten months later, however, the patient presented again with recurrent symptoms.

The patient was taken to the operating room for a

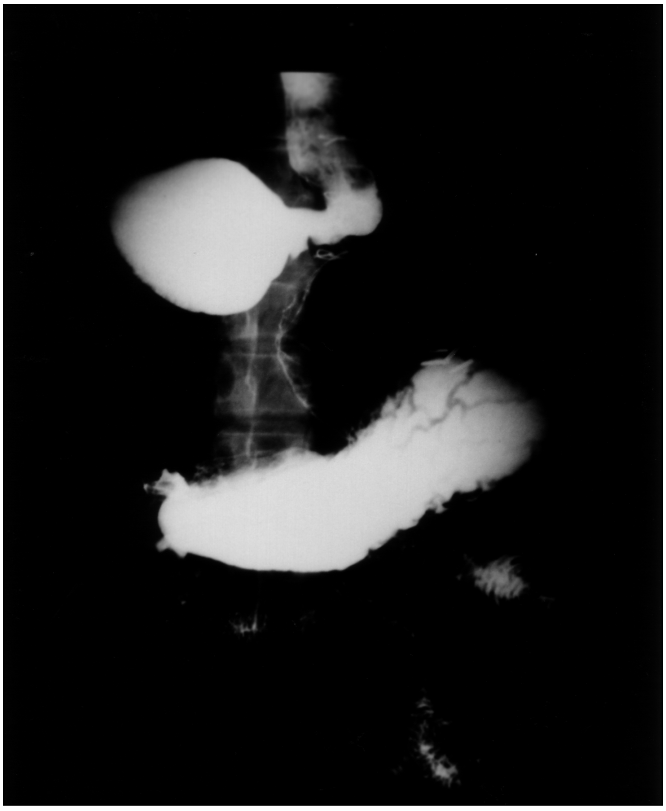

Figure 1. Preoperative barium esophogram picturing the esophageal diverticulum and stenotic LES.

laparoscopic Heller myotomy with resection of the esophageal diverticulum. With the patient in a modified lithotomy position, four 12-mm trocars were placed: one supraumbilically, one at the left anterior axillary line at the level of the umbilicus, and two at the epigastrium approximately 10 cm apart. An additional 5-mm trocar was placed in the right subcostal position for the liver retractor. Harmonic Scalpel® Ultracision® coagulating shears (Ethicon Endosurgery, Inc, Cincinnati, OH) were used to start the dissection through the gastrohepatic ligament to the right crus of the diaphragm. Staying just to the left of the diaphragmatic crus, we continued dissection of the lower esophagus circumferentially in the loose areolar tissue until the diverticulum was identified. The pleural lining of the right chest was separated from the diverticulum laterally, taking care not to enter the right hemithorax. When the diverticulum was completely free of its surrounding attachments, approximately 8 cm of distal esophagus was dissected within the lower mediastinum **(Figure 2)**. An endoscope was then advanced through the mouth and esophagus until the diverticular opening was identified clearly. Endoluminal insufflation from the endoscope assisted in completing the diverticular dissection. A 35-mm Endopath® stapler (Ethicon Endosurgery, Inc, Cincinnati, OH) was used to resect the diverticulum at its base with direct endoluminal visualization to assure no narrowing of the esophagus. The suture line was then reinforced with #0 Ethibond Excel® (Ethicon Endosurgery, Inc, Cincinnati, OH) sutures. The endoscopic balloon dilator was then placed traversing the lower esophageal sphincter. Inflation of the balloon clearly identified the stenotic segment of the esophagus located at the lower esophageal sphincter. The myotomy was performed with the Ultracision® LaparoSonic® (Ethicon Endosurgery, Inc, Cincinnati, OH) dissecting hook. Deflating and inflating the balloon dilator facilitated the dissection by accentuating the partially disrupted muscular fibers. When completed, the myotomy extended 6 cm proximal to the LES and 2 cm distally, until the gastric oblique muscular fibers were encountered. The endoscope was then reinserted and clearly demonstrated a widely patent GE junction. The myotomy had been completed with no apparent injury to the esophageal mucosa.

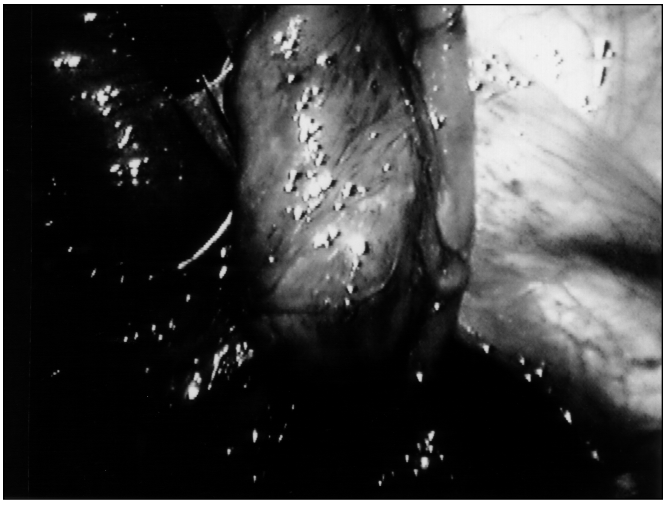

Figure 2. Esophageal diverticulum extending toward the right hemithorax.

The short gastric vessels of the fundus were divided along the fundus up to the left diaphragmatic crus, once

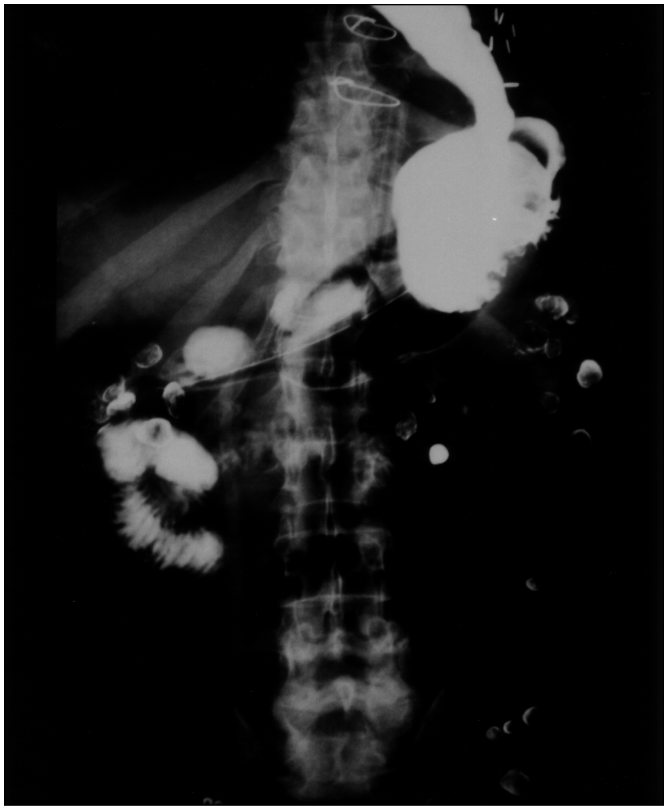

Figure 3. Postoperative barium esophogram showing widely patent LES and no extravasation from diverticulectomy site.

again with the coagulating shears. The fundus was then passed posteriorly around the esophagus. A 2-cm Toupet fundoplication was then constructed by sewing the edges of the wrap to the edges of the myotomy with a #0 Ethibond Excel® suture. The wrap was then secured to the right and left crus of the diaphragm superiorly. The esophagus and stomach were then insufflated while the Toupet fundoplication and protruding esophageal mucosa were held under saline irrigation. No bubbles were seen, indicating that the esophageal mucosa was not injured. The total operative time was 3 hours.

On postoperative day 1, a barium swallow demonstrated that the esophagus was widely patent and had no leakage from either the diverticulectomy suture line or the myotomy site **(Figure 3)**. The patient was discharged on postoperative day 2 on a full liquid diet. Five weeks later, he was eating a regular diet without dysphagia or symptomatic reflux. The patient is currently 17 months status-post Heller myotomy with no dysphagia or symptomatic reflux.

DISCUSSION

Achalasia is not commonly associated with esophageal diverticuli; the reported incidence ranges from 9% to 15% in achalasia patients.[4-6] Investigators have studied why few people with motor disturbances develop lower esophageal diverticuli. Comparing subgroups of patients with and without diverticula within dysmotility groups found no statistical differences between the 2 subgroups.[5,6] Debas et al[5] have suggested that an additional factor, such as weakness in the muscle wall of the esophagus, is necessary for development of the diverticulum.

The myenteric plexus can be likened to a switchboard coordinating central control (via the vagus nerve) and local controls (esophageal dilatation) modulating the LES. LES relaxation is induced by nitrogenic neurons, which release nitric oxide, causing smooth muscle relaxation. Pathologic examination has revealed a paucity of nitrogenic neurons and a mononuclear infiltrate.[7] This finding has led some to believe that achalasia is an autoimmune disease.[8] With the progressive loss of nitric oxide, the LES is under unopposed stimulation via the cholinergic neurons resulting in continuous contraction of the LES. This explains why smooth muscle relaxants like nifedipine and nitrates can provide temporary symptomatic relief in the early stages of achalasia but eventually fail to provide sustained relief.

Physiologically, diverticular formation may be explained because achalasia is characterized by failure of relaxation at the LES creating a high intraluminal pressure. However, classic achalasia presents without esophageal body peristalsis.[8] Olsen et al[9] described a variant of achalasia that exists called "vigorous achalasia." This variant is associated with esophageal body contractions that are normal or even elevated. It can be differentiated from diffuse esophageal spasm because the peristaltic waves are normal and the LES does not relax. It does seem that the conditions created in vigorous achalasia are more conducive to diverticulum formation, yet epiphrenic diverticula can be found associated with LES contraction and no body peristalsis. Some investigators have suggested that vigorous achalasia can be found along the progression to classic achalasia but this is not completely understood.[10]

Multiple studies have demonstrated that asymptomatic epiphrenic esophageal diverticula do not need to be resected.[5,11-13] The difficulty arises in trying to decide whether patients are symptomatic when concurrent abnormalities are discovered. In most cases, patients are symptomatic from their motility or reflux disorder, which is why most patients with mild symptoms can be managed with biennial observation and barium esophagram.[14] In 1993, Benacci et al[11] reported 112 patients with epiphrenic diverticuli. Seventy-one patients had minimal symptoms. Their follow-up is limited to only 35 patients at a mean of 9 years. None of these patients experienced progression of their symptoms.

When performing a laparoscopic Heller myotomy, it is our practice to routinely add an antireflux procedure. The hiatal dissection leads to a dysfunctional gastroesophageal junction. In addition, patients with achalasia have very little, if any, contraction in the esophageal body. Therefore, we choose the Toupet fundoplication because of its demonstrated antireflux capability and low incidence of dysphagia when faced with decreased esophageal motility.[15-17] The reflux rate, defined by symptoms or with the assistance of the pH probe for patients status post-Heller myotomy without an antireflux procedure, ranges from 14% to 57%[18,19] Wang et al[19] reviewed 30 patients with laparoscopic Heller myotomy and found that 14% had frequent regurgitation and 11% reported significant heartburn. Hunter et al[20] reported a series of 40 patients over a 4-year period. Thirty-two had Toupet fundoplication, 7 had Dor fundoplication, and 1 patient had no wrap. This resulted in relief of dysphagia in 90% of patients and elimination of reflux in 95% of patients. Raiser et al[21] reported 10 patients with Dor fundoplication and 29 with Toupet fundoplication in conjunction with the Heller myotomy. Postoperative pH monitoring identified no reflux; however, some patients had heartburn-like symptoms. These symptoms were more common in the Dor fundoplication group.

Our use of the fundoplication has the added benefit of decreasing postoperative reflux with no difference in recurrent dysphagia. Some surgeons choose to use Dor fundoplication because of the perceived benefit of sealing the exposed esophageal mucosa in the event of a leak. We detect esophageal mucosal leaking by insufflating the myotomy site under saline. Perforations are then repaired intraoperatively. We perceive the Toupet to be a better fundoplication when combined with a Heller myotomy because the edges of the myotomy are sutured to the edges of the wrap, which helps keep the edges of the myotomy apart. In addition, we find intraoperative endoscopy to be beneficial because it clearly defines the extent of our GE junction and aids in dissection of the muscular fibers. The benefit of intraoperative endoscopy is further supported by a discordant identification of the GE junction in 58% of patients who had both preoperative and intraoperative endoscopy.[22]

Although this is not the first report of a laparoscopic Heller myotomy with epiphrenic diverticulectomy, our search of the world literature identified 3 other published reports of this approach,[23-25] all with good-to-excellent results. Based on our results and those of others, we believe that laparoscopic Heller myotomy with epiphrenic diverticulectomy can be performed effectively and safely.

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